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Office of Administrative Law Judges
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Issue Date: 30 March 2007

In the Matter of:

M.W., on Behalf of B.W.,
Claimant

Case No.: 2004-BLA-6571

v.

ISLAND CREEK COAL CO.,
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest

APPEARANCES:

Anthony J. Kovach, Esq.
Kovach & Kovach
Uniontown, Pennsylvania
For the Claimant

Ashley M. Harmon, Esq.
Jackson Kelly PLLC
Morgantown, West Virginia
For the Employer

Before: Alice M. Craft
Administrative Law Judge

DECISION AND ORDER AWARDING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901, *et seq*. The Act and implementing regulations, 20 CFR Parts 410, 718, 725, and 727, provide compensation and other benefits to living coal miners, who are totally disabled due to pneumoconiosis, and their dependents, and surviving dependents of coal miners whose death was due to pneumoconiosis. The Act and regulations define pneumoconiosis, commonly known as black lung disease, as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b); 20 CFR § 718.201 (2006). In this case, the Claimant is pursuing a claim on behalf of

her deceased ex-husband, the Miner. She alleges that the Miner was totally disabled by pneumoconiosis.

This claim was set for hearing on April 12, 2005. However, the Miner died on April 10, 2005. I issued an order to show cause why the claim should not be remanded to the District Director to be consolidated with any claim by a survivor. The Claimant and the Employer represented that remand would not be necessary, and the claims could proceed independently. By order dated July 8, 2005, the Miner's former wife, who lived with the Miner and was claimed as his dependent (Director's Exhibits ("DX") 3 and 4) was substituted as the Claimant in order to pursue the claim on his behalf. All parties were afforded a full opportunity to present evidence and argument, as provided in the Rules of Practice and Procedure before the Office of Administrative Law Judges, 29 CFR Part 18 (2006). The parties agreed to submit the case on the record, without the necessity of a hearing. The parties agreed on a schedule to submit additional evidence and closing arguments. No objections have been filed to any of the exhibits. DX 1-43, Claimant's Exhibits ("CX") 1-7, and Employer's Exhibits ("EX") 1-9 are admitted into evidence. The Claimant and the Employer submitted closing arguments, and the record is now closed.

In reaching my decision, I have reviewed and considered the entire record, including all exhibits and the arguments of the parties.

PROCEDURAL HISTORY

The Miner filed his initial claim on April 6, 1991 (DX 1). The claim was denied by Administrative Law Judge Robert G. Mahony on September 15, 1995, who found that the Miner failed to establish the existence of pneumoconiosis. The Miner appealed, and the Benefits Review Board affirmed Judge Mahony's decision on May 31, 1996. The Miner did not pursue the claim further.

More than one year later, the Claimant filed the current claim on May 29, 2003 (DX 3). The Director, OWCP, issued a Proposed Decision and Order awarding benefits on March 31, 2004 (DX 36). The Employer appealed and the claim was forwarded to the Office of Administrative Law Judges on July 22, 2004 (DX 41).

APPLICABLE STANDARDS

This claim relates to a "subsequent" claim filed on May 29, 2003. Because the claim at issue was filed after March 31, 1980, and after January 19, 2001, the effective date of the current regulations, the current regulations at 20 CFR Parts 718 and 725 apply. 20 CFR §§ 718.2 and 725.2 (2006). Pursuant to 20 CFR § 725.309(d) (2006), in order to establish that the Miner was entitled to benefits, the Claimant must demonstrate that "one of the applicable conditions of entitlement … has changed since the date upon which the order denying the prior claim became final" such that the Miner met the requirements for entitlement to benefits under 20 CFR Part 718. In order to establish entitlement to benefits under Part 718, the Claimant must establish that the Miner suffered from pneumoconiosis, that his pneumoconiosis arose out of his coal mine employment, and that his pneumoconiosis was totally disabling. 20 CFR §§ 718.1, 718.202, 718.203, and 718.204 (2006). I must consider the new evidence and determine whether the Claimant has proved at least one of the elements of entitlement previously decided against the Miner. If so, then I must consider whether all of the evidence establishes that he was entitled to

benefits. *Labelle Processing Company v. Swarrow*, 72 F.3d 308 (3rd Cir. 1996); *Lisa Lee Mines v. Director, OWCP*, 86 F.3d 1358 (4th Cir. 1996); *Sharondale Corp. v. Ross*, 42 F.3d 993 (6th Cir. 1994).

ISSUES

The issues contested by the Employer are:

1. The length of coal mine employment by the Miner.

2. Whether the Miner had pneumoconiosis as defined by the Act and the regulations.

3. Whether his pneumoconiosis arose out of coal mine employment.

4. Whether his disability was due to pneumoconiosis.

5. Whether the evidence establishes that one of the applicable conditions of entitlement has changed pursuant to 20 CFR § 725.309 (2006).

The Employer also reserved its right to challenge the statute and regulations (DX 21, 23, 27). Whether the Miner was totally disabled was listed as an issue on the CM 1025 (DX 41), but the Employer is barred from raising this issue because it conceded that the Miner had a total pulmonary disability at the hearing and in its post-hearing brief in the Miner's prior claim (DX 1). *See* Judge Mahony's Decision and Order Denying Benefits at 2. By letter dated August 26, 2006, the Employer stated that the District Director erred in omitting the timeliness of the claim as an issue from the CM-1025; however, the issue of timeliness was not raised in the Employer's Controversion (DX 21), nor in its appeal from the District Director's decision (DX 37), nor in its closing brief. The Employer therefore waived this issue.[1]

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background

The Miner was born in 1931 and died in 2005 (DX 3). He completed the sixth grade (DX 3). The Miner had one dependent for purposes of augmentation of benefits, his former wife, who has been substituted as the Claimant in this claim (DX 3; DX 9).

The Miner's application for benefits lists 45 years of coal mine employment (DX 3). The Miner's Employment History form lists coal mine employment from 1948-1950 and from 1957-1993 (DX 5). The Miner's Social Security earnings worksheet lists coal mine employment from 1949-1993 (CX 7). Based on Social Security earnings I find that the Claimant has established that the Miner had at least 38 years of coal mine employment. On his Employment History, the Miner listed his last coal mine employment as a truck driver (DX 6). The West Virginia

[1] In any event, there is no evidence in the record that any doctor told the Miner that he was totally disabled by pneumoconiosis. Thus there is no evidence that this subsequent claim was not timely filed. *See Westmoreland Coal Co. v. Amick*, 2004 WL 2791653, *3 (4th Cir. 2004) (unpub.); *Wyoming Fuel Co. v. Director, OWCP*, 90 F.3d 1502, 1507 (10th Cir. 1996).

Occupational Pneumoconiosis Board awarded the Miner a 30% pulmonary functional impairment attributable to a diagnosis of occupational pneumoconiosis in 1984, increased to 40% in 1987, while the Miner was still employed in the coal mines (DX 1 (DX 17)).

The Miner's statements about his smoking history varied from 5 years (in the current claim), to 10 to 15 years (in the prior claim), but he consistently reported smoking only a few small cigars or one bowlful of pipe tobacco in a day. I accept the testimony of one of the doctors, who said that the small cigars described by the Miner are essentially equivalent to cigarettes. I find that the Miner smoked for at least 10 years, but had less than a 5 pack year smoking history based on the amount he smoked each day.

The Miner's last full year of coal mine employment was in West Virginia (DX 3). Therefore, this claim is governed by the law of the Fourth Circuit. *Shupe v. Director, OWCP*, 12 B.L.R. 1-200, 1-202 (1989) (*en banc*).

Material Change in Conditions

In a subsequent claim, the threshold issue is whether one of the applicable conditions of entitlement has changed since the previous claim was denied. The Miner's previous claim was denied on the ground that he failed to demonstrate the existence of pneumoconiosis. Absent a finding that he suffered from pneumoconiosis, his claim must fail. Nonrespiratory and nonpulmonary impairments are irrelevant to establishing total disability for the purpose of entitlement to black lung benefits. 20 CFR § 718.204(a) (2006); *Jewell Smokeless Coal Corp. v. Street*, 42 F.3d 241 (4th Cir. 1994); *Beatty v. Danri Corp*., 16 B.L.R. 1-11, 1-15 (1991), *aff'd*. 49 F.3d 993 (3rd Cir. 1995). As will be discussed in detail below, the x-ray and medical opinion evidence filed in connection with the Miner's current claim establishes that the Miner had pneumoconiosis. This constitutes a change in one of the applicable conditions of entitlement. Because the new evidence establishes that a material change in conditions has occurred, I must consider all of the evidence in the record in reaching my decision whether he is now entitled to benefits. Evidence admitted in the prior claim may be considered notwithstanding the limitations on the introduction of evidence contained in 20 CFR § 725.414 (2006). 20 CFR § 725.309(d)(1) (2006). Moreover, no findings in the prior claim are binding, unless a party fails to contest an issue, or made a stipulation in a prior claim. 20 CFR § 725.309(d)(4) (2006).

Medical Evidence

Chest X-rays

Chest x-rays may reveal opacities in the lungs caused by pneumoconiosis and other diseases. Larger and more numerous opacities result in greater lung impairment. The following table summarizes the x-ray findings available in connection with the current subsequent claim. X-ray interpretations submitted by the parties in connection with the current claim are in accordance with the limitations contained in 20 CFR § 725.414 (2006).

The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. Small opacities (1, 2, or 3) (in ascending order of profusion) may classified as round (p, q, r) or irregular (s, t, u), and may be evidence of "simple pneumoconiosis." Large opacities (greater

than 1 cm) may be classified as A, B, or C, in ascending order of size, and may be evidence of "complicated pneumoconiosis." A chest x-ray classified as category "0," including subcategories 0/-, 0/0, and 0/1, does not constitute evidence of pneumoconiosis. 20 CFR § 718.102(b) (2006). Any such readings are, therefore, included in the "negative" column. X-ray interpretations which make no reference to pneumoconiosis, positive or negative, given in connection with medical treatment or review of an x-ray film solely to determine its quality, are listed in the "silent" column.

Physicians' qualifications appear after their names. Qualifications have been obtained where shown in the record by curriculum vitae or other representations, or if not in the record, by judicial notice of the lists of readers issued by the National Institute of Occupational Safety and Health (NIOSH), and/or the registry of physicians' specialties maintained by the American Board of Medical Specialties.[2] If no qualifications are noted for any of the following physicians, it means that either they have no special qualifications for reading x-rays, or I have been unable to ascertain their qualifications from the record, the NIOSH lists, or the Board of Medical Specialties. Qualifications of physicians are abbreviated as follows: A=NIOSH certified A reader; B=NIOSH certified B reader; BCR=Board-certified in Radiology. Readers who are Board-certified Radiologists and/or B readers are classified as the most qualified. *See Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 145 n. 16 (1987); *Old Ben Coal Co. v. Battram*, 7 F.3d 1273, 1276 n.2 (7th Cir. 1993). B readers need not be radiologists.

Date of X-ray	**Read as Positive for Pneumoconiosis**	**Read as Negative for Pneumoconiosis**	**Silent as to the Presence of Pneumoconiosis**
03/07/02	CX 4 Mital B/BCR 1/2	EX 2 Meyer B/BCR	
06/27/03	DX 16 Benjamin B/BCR 2/1, A	DX 18 Wiot B/BCR EX 2 Meyer B/BCR	DX 17 Navani B/BCR Read for quality only ("Poor")
11/17/03	CX 1 Levine 1/2		
01/06/04		EX 2 Meyer B/BCR EX 3, EX 9 Renn[3] B 2/2	

[2]NIOSH is the federal government agency that certifies physicians for their knowledge of diagnosing pneumoconiosis by means of chest x-rays. Physicians are designated as "A" readers after completing a course in the interpretation of x-rays for pneumoconiosis. Physicians are designated as "B" readers after they have demonstrated expertise in interpreting x-rays for the existence of pneumoconiosis by passing an examination. Historical information about physician qualifications appears on the U.S. Department of Health and Human Services, Comprehensive List of NIOSH Approved A and B Readers, February 2, 2007, found at http://www.oalj.dol.gov/PUBLIC/BLACK_LUNG/REFERENCES/REFERENCE_WORKS/BREAD3_02_07.HTM . Current information about physician qualifications appears on the CDC/NIOSH, NIOSH Certified B Readers List found at http://www.cdc.gov/niosh/topics/chestradiography/breader-list.html. Information about physician board certifications appears on the web-site of the American Board of Medical Specialties, found at http://www.abms.org.

[3] Although Dr. Renn classified the x-ray as 2/2, in his deposition he said it was due to emphysema, not pneumoconiosis (EX 9at 15-16). I have therefore included this reading in the negative column.

In the prior claim, Judge Mahony counted 29 interpretations of 13 x-rays taken between February 1984 and May 1994. Six readings were positive, and 23 were negative. He found the weight of the x-ray evidence to be negative for pneumoconiosis, as few were classified as showing opacities in a profusion of at least 1/0. I have reviewed the readings, and find no reason to disagree with his analysis. Despite the characterization of the x-rays as negative for classified opacities indicating pneumoconiosis, however, I also find that the x-ray evidence was consistent with the diagnosis of emphysema.

Pulmonary Function Studies

Pulmonary function studies are tests performed to measure obstruction in the airways of the lungs and the degree of impairment of pulmonary function. The greater the resistance to the flow of air, the more severe the lung impairment. Tests most often relied upon to establish disability in black lung claims measure forced vital capacity (FVC), forced expiratory volume in one-second (FEV_1), and maximum voluntary ventilation (MVV).

The following chart summarizes the results of the newly submitted pulmonary function studies available with the current claim. Pulmonary function studies submitted by the parties in connection with the current claim are in accordance with the limitations contained in 20 CFR § 725.414 (2006). "Pre" and "post" refer to administration of bronchodilators. If only one figure appears, bronchodilators were not administered. In a "qualifying" pulmonary study, the FEV_1 must be equal to or less than the applicable values set forth in the tables in Appendix B of Part 718, and either the FVC or MVV must be equal to or less than the applicable table value, or the FEV_1/FVC ratio must be 55% or less. 20 CFR § 718.204(b)(2)(i) (2006).

Ex. No. Date Physician	**Age Height[4]**	**FEV_1 Pre-/ Post**	**FVC Pre-/ Post**	**FEV_1/ FVC Pre-/ Post**	**MVV Pre-/ Post**	**Qualify?**	**Physician Impression**
CX 4 03/07/02 Gress	71 64"	1.04	2.88	36.1%	25	Yes	Severe obstructive disease and small airways disease. MVV probably invalid.
DX 14 06/27/03 Sagin	72 64.5"	1.22	3.33	37%	N/A	Yes	Severe obstructive disease. Acceptable per Dr. Kucera, DX 15.
CX 1 11/18/03 Levine	72 63"	0.89	2.83	31.4%	35	Yes	Severe obstructive airway disease.
EX 4 01/06/04 Renn	72 64"	0.90 1.06	2.88 3.09	31% 34%	35 40	Yes	Severe obstructive ventilatory defect.

[4] The fact finder must resolve conflicting heights of the miner recorded on the ventilatory study reports in the claim. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221 (1983). I find the Miner's height to have been 64".

There were seven pulmonary function tests, administered between 1984 and 1994, submitted in connection with the prior claim. Only one, administered by Dr. Schmitt on April 26, 1994, resulted in qualifying values. A later test, administered by Dr. Rasmussen on June 15, 1994, did not result in qualifying values. Thus the weight of the pulmonary function tests in the prior claim did not establish total disability.

Arterial Blood Gas Studies

Blood gas studies are performed to measure the ability of the lungs to oxygenate blood. A defect will manifest itself primarily as a fall in arterial oxygen tension either at rest or during exercise. The blood sample is analyzed for the percentage of oxygen (pO_2) and the percentage of carbon dioxide (pCO_2) in the blood. A lower level of oxygen (O_2) compared to carbon dioxide (CO_2) in the blood indicates a deficiency in the transfer of gases through the alveoli which may leave the miner disabled.

The following chart summarizes the newly submitted arterial blood gas studies available in this case. Arterial blood gas studies submitted by the parties in connection with the current claim are in accordance with the limitations contained in 20 CFR § 725.414 (2006). A "qualifying" arterial gas study yields values which are equal to or less than the applicable values set forth in the tables in Appendix C of Part 718. If the results of a blood gas test at rest do not satisfy Appendix C, then an exercise blood gas test can be offered. Tests with only one figure represent studies at rest only. Exercise studies are not required if medically contraindicated. 20 CFR § 718.105(b) (2006).

Exhibit Number	**Date**	**Physician**	**pCO_2 at rest/ exercise**	**pO_2 at rest/ exercise**	**Qualify?**	**Physician Impression**
CX 4	03/07/02	Gress	33 34	58 58	Yes	Hypoxemia at rest and after limited exercise.
DX 13	06/27/03	Sagin	31.9	53.4	Yes	Moderate resting hypoxemia. Acceptable per Dr. Kucera, DX 15.
EX 4	01/06/04	Renn	32.0	50.0	Yes	Acute alkalosis and hypoxemia

The results of three arterial blood gas studies were submitted in connection with the prior claim. The study administered on May 25, 1993, during the Department of Labor examination, was taken at rest only, as the Miner declined an exercise study. That test did not result in a qualifying value. However, both later studies, administered on August 23, 1993, as part of Dr. Renn's examination, and on June 15, 1994, as part of Dr. Rasmussen's examination, included exercise tests which did produce values qualifying for total disability.

Medical Opinions in the Current Claim

Medical opinions are relevant to the issues of whether the miner had pneumoconiosis, whether the miner was totally disabled, and whether pneumoconiosis caused the miner's disability. A determination of the existence of pneumoconiosis may be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers from pneumoconiosis as defined in § 718.201. 20 CFR §§ 718.202(a)(4) (2006). Thus, even if the x-ray evidence is negative, medical opinions may establish the existence of pneumoconiosis. *Taylor v. Director, OWCP*, 9 B.L.R. 1-22 (1986). The medical opinions must be reasoned and supported by objective medical evidence such as blood gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. 20 CFR § 718.202(a)(4) (2006). Where total disability cannot be established by pulmonary function tests, arterial blood gas studies, or cor pulmonale with right-sided heart failure, or where pulmonary function tests and/or blood gas studies are medically contraindicated, total disability may be, nevertheless, found if a physician, exercising reasoned medical judgment, based on medically acceptable clinical and laboratory diagnostic techniques, concludes that a miner's respiratory or pulmonary condition prevents or prevented the miner from engaging in employment, *i.e.*, performing his usual coal mine work or comparable and gainful work. 20 CFR § 718.204(b)(2)(iv) (2006). With certain specified exceptions not applicable here, the cause or causes of total disability must be established by means of a physician's documented and reasoned report. 20 CFR § 718.204(c)(2) (2006). The record contains the following newly submitted medical opinions.

Dr. Gordon Gress examined the Claimant on March 7, 2002, at the request of his counsel (CX 4). Dr. Gress is board certified in Internal Medicine. Much of his practice consists of chest medicine, and includes many soft coal miners. He also examines miners for the presence or absence of pneumoconiosis, and has been an expert witness in litigation regarding pulmonary asbestosis (CX 2). At his examination of the Miner, Dr. Gress noted approximately 42 years of coal mine employment. The Miner could not recall any exposure to asbestos. Dr. Gress recorded symptoms of cough, dyspnea, and sputum. The Miner reported a smoking history of 5 cigars per day for 10 years. Dr. Gress noted past history of prostate cancer and hypertension. Physical examination showed end inspiratory crackles with no rhonchi, rubs or wheezes. Chest x-ray was interpreted as abnormal, 1/2. He also observed pleural plaques. Dilated pulmonary arteries on x-ray suggested possible pulmonary hypertension, and abnormal EKG were suspicious for possible right ventricular hypertrophy such as is seen with cor pulmonale. Pulmonary function study suggested severe obstructive pulmonary disease and small airways pulmonary disease in a non-cigarette smoker. Arterial blood gas showed hypoxemia. Dr. Gress diagnosed pulmonary asbestosis. He said, "I quizzed him carefully regarding possible past exposure to asbestos. In attempt to obtain a clear cut history of asbestos, exposure should be done again." He opined that the Miner is totally disabled and "his long term prognosis is very poor." He went on to state, "I am well aware that he was occupationally exposed to coal dust, according to his history, however, the entire picture is much more the picture of pulmonary asbestosis."

Dr. Mark Sagin examined the Claimant on June 27, 2003, on behalf of the Department of Labor (DX 12). Dr. Sagin is board certified in Internal Medicine, Pulmonary Disease and Critical Care Medicine. He is also certified in sleep disorders (EX 1). He has no special qualifications or credentials in the interpretation of chest x-rays (DX 1 (DX 32)). Dr. Sagin reported that the Miner had 24 years of coal mine employment as a roof bolter and driver. He reported a personal history of attacks of wheezing, arthritis, prostate cancer, and high blood pressure. He reported a

smoking history of five years from 1972-1975 at a rate of five cigars a day. The Miner reported symptoms of sputum, wheezing, dyspnea, cough, and paroxysmal nocturnal dyspnea. On physical examination, Dr. Sagin noted distant breath sounds and crackles. He interpreted an x-ray as showing hyperinflation, with a interstitial/nodular pattern. Pulmonary function studies showed severe obstructive pulmonary impairment, and arterial blood gas readings showed moderate hypoxemia. The Miner's EKG was normal. Dr. Sagin diagnosed chronic obstructive lung disease on the basis of symptoms, physical examination and pulmonary function study. He diagnosed coal workers' pneumoconiosis/silicosis by x-ray, by physical examination, and by the Miner's history of coal dust exposure. He noted that the Miner had a minimal past smoking history and opined that coal and rock dust exposure as an underground miner was the cause of both of the Claimant's pulmonary impairments. Based on the objective testing performed, he opined that the Miner was severely impaired from returning to his previous coal mine employment as a roof bolter. He attributed 80% of the impairment to chronic obstructive pulmonary disease, and 20% to pneumoconiosis.

Dr. Sagin was deposed by the Employer on March 29, 2004 (EX 1). Dr. Sagin said that the Miner's history of coal mine employment was sufficient to cause pneumoconiosis in a susceptible individual. He testified that coal dust exposure generally results in a restrictive impairment of the lungs and not an obstructive impairment. He opined that the Miner's minimal smoking history would not normally cause the degree of obstructive airways disease seen in this case. He testified that this type of severe obstructive pattern generally is not associated with coal dust exposure. He was not sure what caused the Miner's obstructive disease. He opined that the Miner's dust exposure was a minor contribution to severe obstructive airways disease. He opined that any impairment the Claimant derived from his coal workers' pneumoconiosis was minimal or mild. The majority of the Claimant's impairment, in his opinion, resulted from severe obstructive airways disease, which he did not attribute to work in the coal mines. He based that judgment on studies showing the extent to which coal dust contributes to airways obstruction. He said that coal dust exposure does not cause emphysema.

Dr. Sagin also examined the Miner and was deposed in connection with the prior claim. His earlier examination report and deposition testimony are described below.

Dr. Macy Levine examined the Miner at the request of his counsel on November 17, 2003 (CX 1). Dr. Levine is board certified in Internal Medicine and Allergy and Immunology (CX 2). He was a consultant in a chest disease clinic for about 40 years, where most of the patients were coal miners. He noted that the Miner had 45 years of coal mine employment, the last 12 years as a truck driver. He noted past history of hypertension and a smoking history of five years, averaging two cigars a day. On physical examination, he noted that the chest was symmetrical with limited expansion, no resonance and clear breath sounds. An exercise tolerance test consisting of walking over a one step stool was abnormal, manifested by readily seen shortness of breath. Pulmonary function testing indicated the presence of severe obstructive airway disease. Review of a chest x-ray obtained from the University of Pittsburgh Medical Center, showed faint nodular shadows in the lower half of each lung compatible with pneumoconiosis, category 1/2 p/q, in four zones. He diagnosed pneumoconiosis based on a 45 year history of coal dust exposure and an abnormal x-ray. He opined that physical examination and pulmonary function testing showed that the Miner is totally disabled from returning to his previous coal mine job as a truck driver.

Dr. Levine was deposed on September 8, 2004, where he repeated the findings of his earlier report (CX 3; EX 6). He said that the Miner was on home oxygen and other medications for his breathing at the time of the examination. He said the Miner's short history of cigar smoking was not sufficient to produce the changes that he observed. He said that the Miner did not give any history of exposure to asbestos. He said there was no basis to conclude that the Miner had asbestosis because he did not have any of the usual features associated with it, such as pleural plaques or thickening. He said there was no sign of restrictive disease from the pulmonary function test. He said that the degree of obstruction he saw during the Miner's examination was commonly seen with coal dust exposure. He said simple pneumoconiosis typically can cause disabling pulmonary disease. He did not think the Miner had complicated pneumoconiosis. He said he would not diagnose pneumoconiosis absent an abnormality on x-ray. He agreed that not every miner who has x-ray evidence of pneumoconiosis has an associated impairment. He said there was nothing his the Miner's history or physical examination to suggest a cardiac basis for the Miner's shortness of breath. He said the abnormalities on the Miner's x-ray and ventilatory abnormalities went together, and there was no other explanation than pneumoconiosis.

Dr. Joseph J. Renn, a Board-certified Internist, Pulmonologist and B reader, examined the Claimant on January 6, 2004 (EX 4). Dr. Renn also reviewed the Miner's medical records from both claims, as well as the West Virginia Occupational Pneumoconiosis Board Findings from 1984 and 1987. He correctly recited the miner's coal mine employment and noted his last job as a truck driver. The Miner complained of exertional dyspnea, cough, sputum, and wheezing. Dr. Renn noted a five-year smoking history of three to four cigars per day for five years quitting in 1977, a bowlful of pipe tobacco daily, and no smokeless tobacco. However, Dr. Renn noted that at a previous examination in 1993, the Miner had given a 10-year history of cigar smoking, and use of half a package of chewing tobacco daily. On physical examination, the miner's lungs were clear to palpitations and percussion; on auscultation, the Miner had diminished breath sounds and fine expiratory wheezes. There was central and peripheral cyanosis. EKG was normal. Chest x-ray was interpreted as 2/2. Pulmonary function study revealed a severe obstructive ventilatory defect that did not improve following bronchodilator. The resting arterial blood gas test revealed acute respiratory alkalosis and hypoxemia. The carboxyhemoglobin level was consistent with an individual who is not smoking. Dr. Renn diagnosed no pneumoconiosis. He opined that the miner suffers from pulmonary emphysema, and either idiopathic interstitial pulmonary fibrosis or compression of the mid and lower lung zones by emphysema and/or an element of both chronic bronchitis due to tobacco smoking, inadequately controlled systemic hypertension, and arthritis. He opined that the miner would be incapable of performing either his last known coal mining job or any job requiring similar work effort. He opined that none of the miner's diagnoses were caused by or contributed to, by his exposure to coal mine dust.

Dr. Renn was deposed for this claim on July 21, 2005, where he repeated the findings of his earlier written report (EX 9). Dr. Renn said that the Miner had a sufficient history of coal dust exposure to cause coal dust-induced pulmonary disease in a susceptible individual. He noted that the Miner reported no other significant occupational exposures during his working career. Dr. Renn opined that the Claimant's pulmonary capacity diminished considerably from the first test in 1993 to the current test in 2004. He opined that the miner did not suffer from idiopathic pulmonary fibrosis because he did not have a restrictive defect. He stated that hyperinflation, air-trapping, marked reduction in diffusing capacity, and the obstructive nature of the defect is consistent with emphysema caused by smoking, and not with pneumoconiosis. He

believed the Miner had underreported his use of tobacco, as his cotinine levels, which remain elevated longer that nicotine levels, were elevated at the time of his 2004 examination. He opined that the 2/2 opacities he observed on x-ray were found in the mid to lower lung zones, which is inconsistent with pneumoconiosis, which generally starts in the upper zones. He said that emphysema as severe as the Miner's, the chest can compress the lung, causing marking which appear like irregular opacities in the mid and lower zones. He attributed all defects to smoking, and stated that the Miner offered different and varied smoking histories which made it difficult to know his actual smoke exposure level.

Dr. Renn also examined the Miner and was deposed in connection with the prior claim. His earlier examination report and deposition testimony are described below.

Dr. Gregory Fino, a Board-certified Internist, Pulmonologist and B reader, reviewed his own x-ray report and deposition from the Miner's prior claim, the Miner's medical records from the current claim, at the request of the Employer and submitted a report dated May 15, 2005 (EX 7). He opined that x-ray evidence showed pulmonary hypertension and bullous emphysema which progressed between 2002 and 2004. He diagnosed no pneumoconiosis and stated that the emphysema viewed was typical of individuals who had never been exposed to coal mine dust. He opined that pulmonary function testing showed that the Miner is totally disabled from returning to his previous coal mine employment, and that the disability was unrelated to coal mine employment.

Dr. Fino also reviewed the Miner's records and was deposed in connection with the prior claim. His earlier report and deposition testimony are described below.

Medical Opinions in the Prior Claim

The following medical opinions were given in connection with the Miner's prior claim. All are found in DX 1.

Dr. Sagin examined the Claimant on behalf of the Department of Labor on May 25, 1993. He took occupational, social, family and medical histories, and conducted a physical examination, chest x-ray, blood gas studies and pulmonary function testing. The Miner's Employment History form attached to the report indicated that the Miner worked in the mines from 1948 to 1950, and 1956 to 1993. Dr. Sagin reported a smoking history of two to three small cigars per day for 15 years, quitting 10 years before. The chest examination was normal. Dr. Sagin read the x-ray as showing small scattered nodular densities in the upper lung fields, and interstitial pattern predominantly in the lower lung fields. The pulmonary function test showed moderate obstructive disease, but was later found to be invalid. A second test performed July 8, 1993, did not produce qualifying values. The arterial blood gas study revealed hypoxemia and respiratory alkalosis at rest. The Miner declined an exercise test. Dr. Sagin diagnosed chronic obstructive lung disease with chronic bronchitis and industrial bronchitis, based on the pulmonary function test, and coal workers' pneumoconiosis with an element of silicosis based on the chest x-ray. As to the etiology, he said that there was some contribution to the obstructive disease from smoking, although the Miner had only a modest history of smoking. Dr. Sagin presumed significant industrial bronchitis from coal and rock dust exposure. He identified the etiology of the coal workers' pneumoconiosis as the Miner's work exposure, including his time as a roof bolter without respiratory protection, which was a risk factor for

silicosis. He said the Miner was moderately to severely impaired based on his history and pulmonary function tests. He attributed 100% of the Miners impairment to his diagnoses.

In a deposition taken on December 7, 1993, Dr. Sagin testified regarding his examination of the miner. He reiterated the opinion he gave at the time of the examination. He was asked about his understanding of the difference between medical and legal definitions of coal workers' pneumoconiosis. He agreed that the medical definition requires appreciable opacities on x-ray, while the legal definition encompasses any disease which might be caused by exposure to coal dust. He said there is good epidemiologic evidence that there is an association between dust exposure of many types, including coal dust exposure, and airways obstruction. He said the most common cause of chronic obstructive pulmonary disease is tobacco smoking. He agreed that the Miner's 40 years of coal mine employment was sufficient exposure to cause coal workers' pneumoconiosis, but would not necessarily cause pneumoconiosis in all individuals. He did not attach any significance to the resting blood gas which was only minimally abnormal. He characterized the pulmonary function test as moderately abnormal, and thought it to be a valid study. He said the pattern if impairment was similar to that associated with tobacco smoking. He said it is impossible to distinguish between the factors of smoking and dust exposure other than by judgment. He said the Miner was moderately impaired by his pulmonary disease, and whether it would prohibit his returning to coal mine work would depend on what he was doing in the mines. Dr. Sagin said he had made two diagnoses: based on the x-ray and exposure history, either coal worker's pneumoconiosis or silicosis; and based on history and chronic obstructive lung disease, he thought there was a contribution from coal dust exposure. He also thought it possible that the pulmonary impairment arose from tobacco smoking. He could not apportion the contribution of each to the impairment.

Dr. Renn examined the Miner on behalf of the Employer on August 23, 1993. He took occupational, social, family and medical histories, and conducted a physical examination, chest x-ray, blood gas studies and pulmonary function testing. He reported that the Claimant worked in the mines from 1948 to 1993, except for the years 1950 to 1953. He reported no asbestos exposure. He reported a smoking history of three to four little cigars from 1973 to 1983, when he quit all smoking. The chest examination was normal. Dr. Renn read the x-ray as showing small irregular opacities, profusion 1/1, and emphysema. The pulmonary function test showed mild obstructive defect that did not improve after bronchodilator. There was hyperinflation and air trapping. The arterial blood gas study revealed alveolar hyperventilation and excess oxygen tension at rest, which diminished with exercise. Carboxyhemoglobin was normal. Dr. Renn's diagnoses included idiopathic interstitial pulmonary fibrosis and pulmonary emphysema. He said the Miner did not have pneumoconiosis. He said the Miner's moderately severe ventilatory impairment would prevent the Miner from being able to perform his last coal mine job. Dr. Renn said neither of the lung diagnoses was related to coal mine dust. The specific etiology for the pulmonary fibroses and emphysema was uncertain without further diagnostic studies, including a biopsy.

Dr. Renn was deposed on March 10, 1995. He reiterated his findings from his examination of the Miner. He said he was able to exclude coal dust as the cause of the Miner's pulmonary impairment, even though he could not pinpoint its etiology, because of the Miner's symptoms, physical findings and physiologic characteristics. He said the Miner's 10 year history of smoking three to four little cigars from 1973 to 1983 was sufficient a smoking history to produce some of the diseases which can be caused by tobacco smoking such as emphysema,

which showed on his chest x-ray. He also had small irregular opacities characteristic of many different types of interstitial lung disease, but not the small rounded opacities of coal workers' pneumoconiosis. The Miner's obstructive disease was not characteristic of pneumoconiosis. Other possible diagnoses would need a lung biopsy for further delineation. Based on the results of the Miner's pulmonary function studies and arterial blood gas studies, Dr. Renn confirmed that the Miner would not have the ability to perform manual labor. Based on comparison with the 1984 test results, he said that the Miner had mild obstructive disease then, which had progressed over time as a result of aging and pulmonary emphysema. As the disease progresses, an individual would develop exercise-induced hypoxemia, and eventually, resting hypoxemia. He said that studies have not shown a clinically significant decline due to obstructive disease in miners. In Dr. Renn's opinion, the Miner did not have any disease that arose from his exposure to coal mine dust.

Dr. W.K.C. Morgan reviewed the Miner's medical records developed in connection with the claim, and prepared two reports dated October 14, 1993, one addressing two x-rays specifically, and the other addressing x-ray, pulmonary function and examination reports, including the examination reports by Dr. Sagin and Dr. Renn. Dr. Morgan practices thoracic and occupational medicine, and has published a considerable number of articles on various lung diseases, including coal workers' pneumoconiosis. Dr. Morgan was in agreement with Dr. Renn's report. In Dr. Morgan's opinion, the Miner was moderately impaired, sufficiently to make it difficult or impossible for him to work at his previous job in the mines. He said that part of the Miner's impairment was obstructive, while the other part related to an abnormality of gas transfer. Dr. Morgan said the gas transfer impairment was the most severe and likely to interfere with the Miner's capacity to work. He said the obstructive impairment was related to smoking. The gas transfer impairment he thought was probably related to interstitial fibrosis, the cause of which was uncertain. He said the fine irregular opacities in the mid and lower zones seen on x-ray would not be seen in coal workers' pneumoconiosis, but would be seen in smokers or persons with interstitial fibrosis. He also thought the Miner's were partly due to overexpansion and probable emphysema compressing the lower zones, and increasing the markings. Dr. Morgan noted that Dr. Renn did not hear any crackles, which are usually present with interstitial fibrosis, in the Miner's lungs. Although he could not say for certain whether the Miner's impairment was due to fibrosis or smoking given the limited evidence available to him, he was certain that neither coal workers' pneumoconiosis nor exposure to coal mine dust produced the Miner's impairment. He concluded by saying that the gas transfer impairment was "sufficiently severe that [the Miner] would be either unable or seriously distressed in carrying out his last job in the coal mine."

Dr. Richard Schmitt examined the Miner at the request of his counsel on April 26, 1994. Dr. Schmitt is board-certified in internal medicine, pulmonary disease, and critical care medicine. He took occupational, social, family and medical histories, and conducted a physical examination, chest x-ray, and pulmonary function testing. He reported that the Claimant worked in the mines for 44 years. He reported a smoking history of 10 years. Dr. Schmitt observed that the Miner was short of breath when he entered the examination room. The chest examination revealed markedly diminished breath sounds with expiratory airflow delay. Dr. Schmitt read the x-ray as showing mild to moderate profusion of small nodular interstitial opacities throughout both lungs; he did not classify the opacities. The pulmonary function test showed severe obstruction and reduced diffusion. Dr. Schmitt diagnosed coal workers' pneumoconiosis based on the Miner's prolonged history of exposure to coal dust and the chest x-ray, which he said

showed typical changes of a pneumoconiosis. He did not comment whether the Miner was disabled.

Dr. Donald Rasmussen examined the Miner at the request of his counsel on June 15, 1994. Dr. Rassmussen is board-certified in internal medicine, and was not a B reader at the time of the examination. His curriculum vitae reveals substantial work with coal miners and black lung, including consultation with NIOSH. He took occupational, social, family and medical histories, and conducted a physical examination, chest x-ray, blood gas studies and pulmonary function testing. He reported that the Miner worked in the mines for 40 years. He reported a smoking history of 1-3 small cigars for 10 years. The chest examination revealed markedly reduced breath sounds and prolongation of the expiratory phase with forced respirations. Dr. Rasmussen relied on a reading of a chest x-ray by a radiologist, an A reader, who read the x-ray as showing pneumoconiosis, 1/1. The pulmonary function test severe irreversible obstructive impairment. The arterial blood gas study revealed minimal reduction of oxygen at rest, and exercise, which had to be stopped after only three minutes, resulted in markedly impaired oxygen transfer. Dr. Rasmussen diagnosed coal worker's pneumoconiosis. He said the degree of impairment would render the Miner disabled for any significant employment, including his last coal mine employment. He said the primary risk factor for the impairment was coal mine dust exposure, and that smoking could be a contributing factor.

Dr. Rasmussen was deposed on October 20, 1994. He reiterated the opinion he gave at the time of the examination. He confirmed that he did not have any additional medical records to review at the time of the examination, which could have been helpful. He explained why he attributed the Miner's shortness of breath to lung disease rather than other causes such as heart disease, hypertension or obesity. He said the type of opacities found on the Miner's x-rays were not the typical rounded opacities, but were not unusual for coal miners. He said that the pulmonary function tests revealed severe obstructive but not restrictive disease, and would restrict the Miner from light work. He said the pattern of impairment would be consistent with smoking and coal dust exposure. He said the Miner's hypoxia with exercise was due to an oxygen transfer impairment. He said he based his conclusion that the Miner's lung disease was caused by work in the coal mines on the x-ray, the physiological findings and the occupational history. He said that individuals can have a normal x-ray and abnormal pulmonary capacity, a normal x-ray and abnormal pulmonary capacity, or an abnormal x-ray and normal pulmonary capacity.

Dr. Morgan again reviewed the Miner's medical records and physician opinions given in connection with the claim on behalf of the Employer, and prepared a report dated October 5, 1994. Dr. Morgan disagreed with many of Dr. Sagin's comments during his deposition. He concluded that Dr. Sagin had tried to present an objective review of the etiology of the Miner's obstruction, but had failed to correctly assess the current state of knowledge of the disease, and was incorrect when he stated that irregular opacities on x-ray were compatible with coal workers' pneumoconiosis. He particularly noted Dr. Wiot's eminence in the field of x-ray interpretation, and his view that the Miner's x-rays were not compatible with pneumoconiosis. Dr. Morgan concluded that many of the pulmonary function tests he reviewed were less than adequate, but believed the Miner was suffering from a moderate obstruction which would impair him from arduous work. He also thought the Miner to suffer from a significant reduction in his ability to transfer gas; he thought that to be more likely due to emphysema than interstitial fibrosis. In his opinion, the Miner's emphysema was due to cigarette smoking, and not to coal

dust. He thought the abnormalities seen on chest x-ray suggested idiopathic pulmonary fibrosis, or some other fibrotic condition. He did not believe there was any objective evidence to justify a diagnosis of coal workers' pneumoconiosis.

Dr. Fino reviewed additional medical records on behalf of the Employer, including various pulmonary function studies, x-ray interpretations, the reports of the Miner's various physical examinations, and doctors' depositions, and provided a report dated February 23, 1995. Dr. Fino opined that the Miner did not suffer from an occupationally acquired pulmonary condition as a result of coal mine dust exposure, based on the majority of negative x-ray readings; spirometric evaluations showing a pure obstructive ventilatory impairment with involvement in the small airways, but no restrictive defect; and over-inflated lung volumes demonstrating air-trapping due to obstructive disease, typical for emphysema, asthma, or chronic obstructive bronchitis, but not the contraction of lung tissue due to fibrosis as would be expected in simple coal workers' pneumoconiosis. Dr. Fino said that there was disabling respiratory impairment secondary to emphysema. He said the Miner would be as disabled had he never stepped foot in the coal mines.

Dr. Fino was deposed on March 29, 1995. He confirmed his opinions given in his reports of August 1993 and February 1995. He agreed that the Miner had sufficient exposure to coal dust for a susceptible individual to develop pneumoconiosis. He continued to maintain that the Miner did not have pneumoconiosis. He cited to literature which does not support the conclusion that there is a significant or clinically detectible obstructive lung disease in retired miners. He attributed the Miner's disabling pulmonary disease to emphysema. He did not believe that interstitial fibrosis was present, as some other physicians did. He did not believe that the Miner's respiratory problems could be tied to smoking, either. He said Dr. Renn's testing ruled out $alpha_1$-antitrypsin deficiency, but said there are other forms of hereditary emphysema which might explain the Miner's pattern. He reiterated that he did not believe that the Miner had a chronic dust disease of the lung arising out of coal mine employment, nor did he have any impairment or pulmonary disease related to, substantially aggravated by or caused by dust exposure during his coal mine employment. He did not see any evidence of asbestos disease. He also confirmed that the Miner was disabled from his last coal mine employment.

Existence of Pneumoconiosis

The regulations define pneumoconiosis broadly:

> (a) For the purpose of the Act, 'pneumoconiosis' means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or 'clinical,' pneumoconiosis and statutory, or 'legal,' pneumoconiosis.
>
> > (1) Clinical Pneumoconiosis. 'Clinical pneumoconiosis' consists of those diseases recognized by the medical community as pneumoconioses, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis,

> anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silico-tuberculosis, arising out of coal mine employment.
>
> (2) Legal Pneumoconiosis. 'Legal pneumoconiosis' includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.
>
> (b) For purposes of this section, a disease 'arising out of coal mine employment' includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.
>
> (c) For purposes of this definition, 'pneumoconiosis' is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 CFR § 718.201 (2006). In this case, the Claimant's medical records indicate that he has been diagnosed with chronic obstructive pulmonary disease and emphysema, which can be encompassed within the definition of legal pneumoconiosis. *Ibid*.; *Richardson v. Director, OWCP*, 94 F.3d 164 (4th Cir. 1996); *Warth v. Southern Ohio Coal Co*., 60 F.3d 173 (4th Cir. 1995). However, only chronic obstructive pulmonary disease caused by coal mine dust constitutes legal pneumoconiosis. *Eastover Mining Co. v. Williams*, 338 F.3d 501, 515 (6th Cir. 2003).

Twenty CFR § 718.202(a) (2006) provides that a finding of the existence of pneumoconiosis may be based on: (1) chest x-ray; (2) biopsy or autopsy; (3) application of the presumptions described in §§ 718.304 (irrebuttable presumption of total disability if there is a showing of complicated pneumoconiosis), 718.305 (not applicable to claims filed after January 1, 1982), or 718.306 (applicable only to deceased miners who died on or before March 1, 1978); or, (4) a physician exercising sound medical judgment based on objective medical evidence and supported by a reasoned medical opinion. There is no evidence that the Claimant had a lung biopsy or that an autopsy was performed. As to the presumptions, there is some evidence in the record that the Miner had complicated pneumoconiosis which is addressed below. Neither of the other presumptions apply, because the Miner filed his claim after January 1, 1982, and he died after March 1, 1978. In order to determine whether the evidence establishes the existence of simple or complicated clinical pneumoconiosis, or legal pneumoconiosis, therefore, I must consider the chest x-rays and medical opinions. Absent contrary evidence, evidence relevant to either category may establish the existence of pneumoconiosis. In the face of conflicting evidence, however, I must weigh all of the evidence together in reaching my finding whether the Claimant has established that he has pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 211 (4th Cir. 2000).

Pneumoconiosis is a progressive and irreversible disease. *Labelle Processing Co. v. Swarrow*, 72 F.3d 308, 314-315 (3rd Cir. 1995); *Lane Hollow Coal Co. v. Director, OWCP*, 137 F.3d 799, 803 (4th Cir. 1998); *Woodward v. Director, OWCP*, 991 F.2d 314, 320 (6th Cir. 1993).

As a general rule, therefore, more weight is given to the most recent evidence. *See Mullins Coal Co. of Virginia v. Director, OWCP*, 484 U.S. 135, 151-152 (1987); *Eastern Associated Coal Corp. v. Director, OWCP*, 220 F.3d 250, 258-259 (4th Cir. 2000); *Crace v. Kentland-Elkhorn Coal Corp*., 109 F.3d 1163, 1167 (6th Cir. 1997); *Rochester & Pittsburgh Coal Co. v. Krecota*, 868 F.2d 600, 602 (3rd Cir. 1989); *Stanford v. Director, OWCP*, 7 B.L.R. 1-541, 1-543 (1984); *Tokarcik v. Consolidated Coal Co*., 6 B.L.R. 1-666, 1-668 (1983); *Call v. Director, OWCP*, 2 B.L.R. 1-146, 1-148 to 1-149 (1979). This rule is not to be mechanically applied to require that later evidence be accepted over earlier evidence. *Woodward*, 991 F.2d at 319-320; *Adkins v. Director, OWCP*, 958 F.2d 49 (4th Cir. 1992); *Burns v. Director, OWCP*, 7 B.L.R. 1-597, 1-600 (1984).

Turning first to the x-ray evidence in the current claim, the March 7, 2002, x-ray film was read as positive for simple pneumoconiosis by Dr. Mital, who is a board certified radiologist, according to the American Board of Medical Specialties, and a B reader, according to the NIOSH list. It was read as negative by Dr. Meyer, who is also dually qualified. As both readers are equally qualified, I find this x-ray to be inconclusive.

The June 27, 2003, x-ray was read as negative by Drs. Wiot and Meyer, both Board-certified Radiologists and B readers, and as positive for simple and complicated pneumoconiosis by Dr. Benjamin, who is also dually certified. Noting identical qualifications as to board and NIOSH certification, I give greater weight to the two negative readings over the one positive reading. Moreover, I note that Dr. Wiot is preeminent in the field of interpreting x-rays for the purpose of diagnosing clinical pneumoconiosis, *see* his curriculum vitae (DX 18) and his deposition (DX 1 (DX 41)), which adds additional weight to his negative reading. As Dr. Benjamin was the only radiologist who diagnosed complicated pneumoconiosis based on x-ray, I find that the June 27, 2003, x-ray evidence is negative for either simple or complicated pneumoconiosis.

The November 17, 2003, x-ray was read as positive for simple pneumoconiosis by Dr. Levine. There are no negative readings. I find this x-ray to be positive for simple pneumoconiosis.

The January 6, 2004, x-ray film was interpreted as negative by Dr. Meyer, a dually certified physician, and as negative by Dr. Renn, a B reader, despite a classification of opacities in the lower lungs as 2/2. As the record does not contain a conflicting positive reading, I find that the January 6, 2004, x-ray evidence is negative for pneumoconiosis.

The newly submitted record contains two negative films, one inconclusive film, and one positive film. I find that the preponderance of newly submitted x-ray evidence is not sufficient to form the basis for a finding of pneumoconiosis, but neither does it rule it out.

I must next consider the medical opinions. The Claimant can establish that he suffers from pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's work and social histories. *Hoffman v. B&G Construction Co*., 8 B.L.R. 1-65, 1-66 (1985); *Hess v. Clinchfield Coal Co*., 7 B.L.R. 1-295, 1-

296 (1984); *Justus v. Director, OWCP*, 6 B.L.R. 1-1127, 1-1129 (1984). A "reasoned" opinion is one in which the Judge finds the underlying documentation and data adequate to support the physician's conclusions. *Fields*, above. Whether a medical report is sufficiently documented and reasoned is for the Judge to decide as the finder-of-fact; an unreasoned or undocumented opinion may be given little or no weight. *Clark v. Karst-Robbins Coal Co*., 12 B.L.R. 1-149, 1-155 (1989) (*en banc*).

The Department of Labor has taken the position that coal dust exposure may induce obstructive lung disease even in the absence of fibrosis or complicated pneumoconiosis. This underlying premise was stated explicitly in the commentary that accompanied the final version of the current regulations. The Department concluded that "[e]ven in the absence of smoking, coal mine dust exposure is clearly associated with clinically significant airways obstruction and chronic bronchitis. **The risk is additive with cigarette smoking**." 65 Fed. Reg. at 79940 (emphasis added). Citing to studies and medical literature reviews conducted by NIOSH, the Department quoted the following from NIOSH:

> … COPD may be detected from decrements in certain measures of lung function, especially FEV1 and the ratio of FEV1/FVC. **Decrements in lung function associated with exposure to coal mine dust are severe enough to be disabling in some miners, whether or not pneumoconiosis is also present**.…

65 Fed. Reg. at 79943 (emphasis added). Moreover, the Department concluded that the medical literature "support[s] the theory that dust-induced emphysema and smoke-induced emphysema occur through similar mechanisms." Medical opinions which are based on the premise that coal dust-related obstructive disease is completely distinct from smoking-related disease, or that it is never clinically significant, are therefore contrary to the premises underlying the regulations. I have considered how to weigh the conflicting medical opinions in this case based on these principles.

Dr. Gress diagnosed pulmonary asbestosis. He noted smoking and coal dust exposure histories and stated that "I am well aware that [the Claimant] was occupationally exposed to coal dust, … however, the entire picture [of objective testing] is much more the picture of pulmonary asbestosis [and not pneumoconiosis]." He stated that the Miner's minimal smoking history, when viewed with the reported symptoms, physical examination findings, x-ray interpretation and pulmonary function and arterial blood gas testing, all suggested severe obstructive pulmonary disease with an unknown but possibly asbestos-related causation. Dr. Gress is the only doctor who diagnosed asbestosis, other than Dr. Wiot who suggested that the opacities he observed on x-ray might be related to asbestosis. However, there is no creditable evidence in the record that the Miner was exposed to asbestos during his lifetime. Drs. Levine and Fino, who took opposite positions on whether the Miner had pneumoconiosis, both agreed that he did not have asbestosis. Although Dr. Gress' observations relating to the Miner's physical condition and pulmonary impairment were well documented, I find that his diagnosis was not well-reasoned, and give it little weight.

Dr. Sagin diagnosed simple clinical coal workers' pneumoconiosis based on an abnormal x-ray, physical examination and a history of coal dust exposure. Dr. Sagin also diagnosed chronic obstructive pulmonary disease, based on symptoms, physical examination and pulmonary function study. In his written report, he noted a minimal smoking history and opined

that rock dust exposure as a coal miner was the cause of the Miner's pulmonary impairments. I construe this to be a diagnosis of legal as well as clinical pneumoconiosis. In his deposition, however, he stated that coal dust generally does not cause the obstructive defect seen in the Miner, and he opined that the Miner's severe obstructive airways disease was *not* attributable to working in coal mines. It is proper to accord little probative weight to the opinions of a physician who has given inconsistent opinions without explaining the reasons for the inconsistency. *Hopton v. U.S. Steel Corp.,* 7 B.L.R. 1-12, 1-14 (1984); *Surma v. Rochester & Pittsburgh Coal Co*., 6 B.L.R. 1-799, 1-801-802 (1984). In this case, at his deposition, Dr. Sagin did a complete about-turn from the opinion he gave in his report on the cause of the Miner's pulmonary disease, without any explanation for such a reversal. Moreover, Dr. Sagin testified that he was not aware of any studies associating coal dust exposure with obstructive disease. The commentary to the regulations demonstrates that such literature does exist. Noting Dr. Sagin's inconsistencies on the cause of the Miner's obstructive disease, and his lack of familiarity with any studies linking coal dust exposure to obstructive disease such as those relied upon by NIOSH and the Department of Labor, I give his opinion little weight on the issue of whether the Miner had pulmonary disease related to his coal mine employment.

Dr. Levine diagnosed coal workers' pneumoconiosis based on a history of coal dust exposure and an abnormal x-ray. He read the x-ray taken at the time of his examination to be positive, 1/2, and as there were no negative readings, I have found it to be positive. Moreover, although I have found that the x-ray evidence as a whole was insufficient to form the basis of a finding of pneumoconiosis, other physicians who read the Miner's x-rays also observed abnormalities. Dr. Wiot interpreted the x-ray taken on June 27, 2003, to show chronic obstructive pulmonary disease and emphysema. He also said that there was prominence to the central pulmonary vessels that raised a question of pulmonary hypertension. DX 18. All of the other readers who found no pneumoconiosis also observed abnormalities including emphysema, hyperinflation, possible prior granulomatous disease, and pulmonary hypertension. At his deposition, Dr. Levine considered and rejected various possible causes of the Miner's obstructive disease and shortness of breath, including asbestos exposure, smoking, and heart disease. He concluded that there was no other explanation than coal dust exposure to explain the Miner's severe obstructive disease. I find Dr. Levine's opinion to be well-reasoned and well-documented, and that it supports a finding of clinical and legal pneumoconiosis.

Dr. Renn diagnosed no pneumoconiosis and opined instead that the Miner suffered from pulmonary emphysema. He based his opinion on the obstructive nature of the defect, hyperinflation of the lungs, air-trapping, and a marked reduction in diffusing capacity. He opined that all of these findings were consistent with emphysema due to smoking and inconsistent with pneumoconiosis. He noted that the Miner had offered inconsistent smoking histories over a 10 year period, and he questioned whether the Miner had accurately reported his true smoking history. He also noted that carboxyhemoglobin did confirm that the Miner was not presently smoking, but his cotinine level was elevated. He explained that the 2/2 opacities he observed on x-ray likely represented marking from compression of the lungs due to severe emphysema. Although he initially thought the Miner may have had interstitial fibrosis, in his most recent deposition, he rejected that diagnosis. Dr. Renn has superior credentials, and his report is well documented. However, he appears to reject the premise behind the regulations that exposure to coal dust can cause clinically significant obstructive disease. I have found that the Miner had 38 years of coal mining, and less than a 5 pack year history of smoking. Dr. Renn has failed to provide any convincing reasons for discounting the contribution of coal dust exposure to

the Miner's admittedly severe obstructive disease. I find that Dr. Renn's report is not well reasoned on the cause of the Miner's obstructive disease, and give it little weight.

Dr. Fino diagnosed hypertension and pulmonary emphysema based on his review of the Miner's records, including x-rays, pulmonary function studies, physical examinations, and arterial blood gas studies. Dr. Fino rejected diagnoses of pulmonary fibrosis and asbestosis, suggested by others. Dr. Fino opined that the evidence showed progressive bullous emphysema typical of individuals who had never been exposed to coal mine dust. He opined that the readings presented were inconsistent with coal dust related ailments. Like Dr. Renn's opinion, I find that Dr. Fino's opinion was based on premises contrary to those underlying the current regulations. I also find that Dr. Fino failed to provide any convincing reason for excluding the Miner's 38 years of exposure to coal dust as a factor contributing to his obstructive disease. The fact that the same progression of disease can be found in a smoker who is not a miner, does not mean that coal dust does not contribute to the disease in a miner, especially where, as here, the Miner has a much more substantial history of exposure to coal dust than to smoking. Thus I also find that Dr. Fino's opinion is not well reasoned on the cause of the Miner's obstructive disease.

Taken as a whole, I find that the Claimant has established that the Miner had simple clinical pneumoconiosis, and legal pneumoconiosis, based on the newly submitted x-ray evidence, and the medical opinion of Dr. Levine. The Claimant has thereby established a material change in conditions, and I must also consider the evidence from the Miner's prior claim in reaching my decision whether he was entitled to black lung benefits.

As noted above, the most recent x-rays in the prior claim dated back to 1994, and are therefore remote in time. Despite the negative readings for pneumoconiosis, many of the readers noted other abnormalities in the x-rays. Dr. Wiot was deposed in the prior claim, and testified that the markings on the Miner's chest x-rays he saw, which were irregular in shape rather than rounded, plus a few rounded opacities, would be classified as 0/1, and represented some disease process other than coal workers' pneumoconiosis. He thought the changes on the x-ray were due to some form of pulmonary fibrosis, most likely, idiopathic pulmonary fibrosis, although they were also compatible with asbestosis. However, those alternate diagnoses were rejected by Drs. Levine, Renn and Fino. Dr. Wiot also said that other findings on the x-rays were consistent with emphysema. He said that it would not be fair to say that smoking was the only cause of the emphysema. I conclude that the weight of the x-ray interpretations from the prior claim was negative for clinical pneumoconiosis, but that conclusion has little bearing on the weighing of the x-ray evidence in the current claim due to their remoteness in time. Moreover, although the weight of the x-ray evidence in the prior claim was negative for clinical pneumoconiosis, that evidence also revealed the Miner's progressive obstructive disease. Thus the x-ray evidence from the prior claim does not change my finding that the Miner had pneumoconiosis based on more recent evidence.

Turning to the medical opinions in the prior claim, the fact that Dr. Sagin then believed that coal dust contributed to the Miner's obstructive disease is of little moment due to his later change of position on that point. Two others, Dr. Schmitt and Dr. Rasmussen, also said that the Miner had pneumoconiosis. Dr. Schmitt appeared to rely heavily on the x-ray evidence which I have found to be negative for clinical pneumoconiosis. His opinion is somewhat ambiguous whether he considered both clinical and legal pneumoconiosis. Nonetheless, he also had other evidence available to him, and his opinion is entitled to some weight, and it supports

Dr. Levine's opinion. Dr. Rasmussen, like Dr. Levine, diagnosed both clinical and legal pneumoconiosis. He identified the primary risk factor for the Miner's obstructive impairment to be coal dust exposure, and said that smoking could also be a contributing factor. Dr. Rasmussen also explained why other possible causes of the Miner's symptoms, such as heart disease, hypertension or obesity, could be rejected. Finally, I find that his history of working with miners, and the fact that he was able to examine the Miner in connection with his claim, gave him a solid basis to form an opinion. I find that Dr. Rasmussen's opinion was well documented and well reasoned, and that it supports Dr. Levine's opinion in the current claim.

In the previous claim, Dr. Renn diagnosed idiopathic pulmonary fibrosis and emphysema. In the later claim, however, Dr. Renn rejected the diagnosis of idiopathic pulmonary fibrosis because the Miner did not have a restrictive impairment. His opinion that the Miner suffered from emphysema caused by smoking alone remained consistent over the years. As noted above, however, I have given his opinion on this issue little weight because his opinion that coal dust exposure did not contribute to the Miner's emphysema is not well reasoned.

Dr. Morgan also diagnosed interstitial fibrosis in the first claim, a diagnosis he then shared with Dr. Renn, but which Dr. Renn later ruled out. Moreover, in his deposition, Dr. Morgan said he thought the Miner's gas transfer abnormality to be due to emphysema, and not interstitial fibrosis. Dr. Morgan, like Dr. Renn, said that the Miner's obstructive disease was due to smoking, but offered no convincing reason for ruling out coal dust exposure as a contributing factor.

Similarly, Dr. Fino took consistent positions, not well explained, that coal dust did not contribute to the Miner's emphysema. In the first claim, Dr. Fino also ruled out interstitial fibrosis and asbestosis as possible diagnoses. Thus I find that his opinion, along with Dr. Renn's, supports the conclusion that the Miner did not have either idiopathic pulmonary fibrosis, or asbestosis. Indeed, the fact that both Dr. Fino and Dr. Renn, who are pulmonologists, rejected the diagnoses of pulmonary fibrosis and asbestosis, undermines Dr. Wiot's diagnosis made on the more limited basis of x-ray interpretation alone, as well as Dr. Gress' opinion that the Miner had asbestosis. Although I give some weight to both Dr. Fino's and Dr. Renn's opinions that the Miner did not suffer from pulmonary fibrosis or asbestosis, as those opinions are consistent with the weight of the evidence as a whole, I do not credit their opinions that coal dust did not contribute to the Miner's obstructive disease.

Considering the x-ray and medical opinion evidence from both claims together, I find that the Claimant has established that the Miner had pneumoconiosis.

Causal Relationship Between Pneumoconiosis and Coal Mine Employment

The Act and the regulations provide for a rebuttable presumption that pneumoconiosis arose out of coal mine employment if a miner with pneumoconiosis was employed in the mines for ten or more years. 30 U.S.C. § 921(c)(1); 20 CFR § 718.203(b) (2006). The Miner was employed as a miner for at least 38 years, and therefore is entitled to the presumption. The Employer has not offered evidence sufficient to rebut the presumption. Moreover, to the extent that Claimant has legal, as opposed to clinical pneumoconiosis, the causal relationship is established by the opinions of Dr. Levine and Dr. Rasmussen. I conclude that the Miner's pneumoconiosis was caused by his coal mine employment.

Total Pulmonary or Respiratory Disability

As noted above, the Employer conceded in the Miner's first claim that he was totally disabled. A miner is considered totally disabled if he has complicated pneumoconiosis, 30 U.S.C. § 921(c)(3), 20 CFR § 718.304 (2006), or if he has a pulmonary or respiratory impairment to which pneumoconiosis is a substantially contributing cause, and which prevents him from doing his usual coal mine employment and comparable gainful employment, 30 U.S.C. § 902(f), 20 CFR § 718.204(b) and (c) (2006). I have found that the x-ray evidence did not establish that the Miner had complicated pneumoconiosis, and no doctor has given such a diagnosis, either. The regulations provide five methods to show total disability other than by the presence of complicated pneumoconiosis: (1) pulmonary function studies; (2) blood gas studies; (3) evidence of cor pulmonale; (4) reasoned medical opinion; and (5) lay testimony. 20 CFR § 718.204(b) and (d) (2006). In the prior claim, the exercise blood gas studies and the medical opinion evidence established that the Miner was disabled. In the current claim, the pulmonary function tests, the arterial blood gas studies, and the medical opinions establish that the Miner's impairment progressed after the denial of his first claim, and that he continued to be disabled.

Causation of Total Disability

In order to be entitled to benefits, the Claimant must establish that pneumoconiosis is a "substantially contributing cause" to the Miner's disability. A "substantially contributing cause" is one which has a material adverse effect on the miner's respiratory or pulmonary condition, or one which materially worsens another respiratory or pulmonary impairment unrelated to coal mine employment. 20 CFR § 718.204(c) (2006); *Hobbs v. Clinchfield Coal Co*., 917 F.2d 790, 792 (4th Cir. 1990); *Robinson v. Pickands Mather & Co*., 914 F.2d 35, 38 (4th Cir. 1990).

The current regulations state that unless otherwise provided, the burden of proving a fact rests with the party making the allegation. 20 CFR § 725.103 (2006). The Benefits Review Board has held that Section 718.204 places the burden on the claimant to establish total disability due to pneumoconiosis by a preponderance of the evidence. *Baumgardner v. Director, OWCP*, 11 B.L.R. 1-135 (1986). Nothing in the commentary to the new rules suggests that this burden has changed; indeed, some language in the commentary indicates it has not changed. See 65 Fed. Reg. at 79923 (2000) ("Thus, a miner has established that his pneumoconiosis is a substantially contributing cause of his disability if it either has a material adverse effect on his respiratory or pulmonary condition or materially worsens a totally disabling respiratory or pulmonary impairment …"). The Fourth Circuit requires that pneumoconiosis be a "contributing cause" of the miner's disability. *Hobbs v. Clinchfield Coal Co*., 917 F. 2d 790, 791-792 (4th Cir. 1990). In *Toler v. Eastern Associated Coal Co*., 43 F.3d 109 (4th Cir. 1995), the Court found it "difficult to understand" how an Administrative Law Judge (ALJ), who finds that the claimant has established the existence of pneumoconiosis, could also find that his disability is not due to pneumoconiosis on the strength of the medical opinions of doctors who had concluded that the claimant did not have pneumoconiosis. The Court noted that there was no case law directly in point and stated that it need not decide whether such opinions are "wholly lacking in probative value." However the Court went on to hold:

> Clearly though, such opinions can carry little weight. At the very least, an ALJ who has found (or has assumed arguendo) that a claimant suffers from

> pneumoconiosis and has a total pulmonary disability may not credit a medical opinion that the former did not cause the latter unless the ALJ can and does identify specific and persuasive reasons for concluding that the doctor's judgment on the question of disability does not rest upon her disagreement with the ALJ's finding as to either or both of the predicates in the causal chain.

43 F.3d at 116. *See also Scott v. Mason Coal Company*, 289 F.3d 263, 269-270 (4th Cir. 2002).

In this case, I can find no specific and persuasive reasons for concluding that the judgment of any of the doctors who believed that exposure to coal dust did not cause or contribute to the Miner's disability did not rest upon his disagreement with my finding that the Miner had pneumoconiosis. All of the physicians consulted by the Employer blamed the Miner's obstructive pulmonary condition on his cigarette smoking alone, or to other causes that were later excluded. I have found that the Miner had less than a 5 pack year history of smoking, but a 38 year history of working in the mines. None of the Employer's experts acknowledged that coal dust can contribute to obstructive disease. Their complete dismissal of any effects from exposure to coal dust lead me to the conclusion that their opinions are less than objective. Accordingly, I assign less weight to their opinions than I do to Dr. Levine's and Dr. Rasmussen's.

Date of Entitlement

In the case of a miner who is totally disabled due to pneumoconiosis, benefits commence with the month of onset of total disability. Medical evidence of total disability does not establish the date of entitlement; rather, it shows that a claimant became disabled at some earlier date. *Owens v. Jewell Smokeless Coal Corp.*, 14 BLR 1-47, 1-50 (1990). Where the evidence does not establish the month of onset, benefits begin with the month that the claim was filed, unless the evidence establishes that the miner was not totally disabled due to pneumoconiosis at any subsequent time. 20 CFR § 725.503(b) (2006); *Harris v. Old Ben Coal Co.*, 23 B.L.R. 1-___, BRB No. 04-0812 BLA (Jan. 27, 2006), slip op. at 17.

The Miner filed his claim for benefits on May 29, 2003. His exercise arterial blood gas studies show that he was totally disabled by August 1993. The regulation regarding subsequent claims also provides, however, that "In any case in which a subsequent claim is awarded, no benefits may be paid for any period prior to the date upon which the order denying the prior claim became final." 20 CFR § 725.309(d)(5). The Benefits Review Board affirmed Judge Mahony's decision on May 31, 1996. As the Miner took no further action on that claim, it became final one year later, on May 31, 1997.

I find that the Miner was entitled to benefits commencing in May 1997, the month in which the decision on his prior claim became final.

ENTITLEMENT TO BENEFITS

Having considered all of the relevant evidence, I find that the Claimant has established that the Miner had pneumoconiosis arising out of his coal mine employment, and a totally disabling pulmonary or respiratory impairment caused by pneumoconiosis. Thus the Claimant

has met her burden of showing a change in an applicable condition of entitlement pursuant to § 725.309(d). Accordingly, the Miner was entitled to benefits under the Act.

ATTORNEY FEES

The regulations address attorney's fees at 20 CFR §§ 725.362, 365 and 366 (2006). The Claimant's attorney has not yet filed an application for attorney's fees. The Claimant's attorney is hereby allowed thirty days (30) days to file an application for fees. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. The other parties shall have ten (10) days following service of the application within which to file any objections, plus five (5) days for service by mail, for a total of fifteen (15) days. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claim for benefits filed by the Miner on May 29, 2003, is hereby GRANTED.

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ALICE M. CRAFT
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the Administrative Law Judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the Administrative Law Judge's decision is filed with the District Director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC, 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC, 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the Administrative Law Judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).